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Anti-atherogenic function of LPL in human and porcine coronary endothelial cells

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Multiple epidemiological studies concluded that lipoprotein lipase (LPL) function is inversely related to the incidence and severity of coronary artery disease. There is debate, however, because those seeking to identify the responsible mechanisms have reported higher than normal levels of LPL in the arteries of diseased mouse models. This confusion could be clarified by beginning to identify the LPL responses in pigs as a large animal model and determining the phenotypic effects of experimentally altering LPL on cultured endothelial cells and isolated vascular tissue. PURPOSE: We tested the hypothesis that LPL activity regulates the expression of thrombospondin-1 (TSP-1), eNOS, VCAM1, and the PPAR promoter activity in endothelial cells. METHODS: LPL activity and protein were measured in the plasma and heart of pigs fed a normal or high fat diet. The cellular effects of changing LPL activity were determined in primary porcine and human endothelial cells. Studies of VCAM1 were performed in cultured cells and isolated aortic segments. Northern and Western blots were used for mRNA and protein measurements, respectively. RESULTS: LPL-dependent lipolysis of VLDL suppressed TSP-1 expression several fold in endothelial cells (P

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